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AN ANATOMICAL AND BACTERIOLOGICAL STUDY OF ACUTE DIFFUSE NEPHRITIS.

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The kidney is a glandular organ, but there is no gland in the body with which it may be compared. The peculiarity in the structure of the kidney lies in its blood-supply and in its structure as a gland. There is in the kidney a structure corresponding to a gland, and leading from this an excretory duct which also has true glandular properties of its own, so that the kidney may be considered as a double glandular organ. The glomeruli have the structure of true glands, with capillary wall, basement-membrane, and differentiated epithelium. The tubule leading from the glomerulus may be considered partly as an excretory duct for that glandular structure, but it has glandular functions of its own as well, and adds various substances to the secretions of the glomerulus. The circulation of the kidney is peculiar in that nearly all of the blood passes first to the glomerulus, and after circulating in the capillaries of this leaves the glomerulus in a single vessel, which again breaks up into capillaries which are distributed between the tubules. From this it can be seen that any lesions of the glomerulus which in any way interfere with the circulation of the blood in its vessels must exert a marked action on the nutrition and function of the other parts. The glomeruli, tubules, and vessels are held together by delicate interstitial tissue in which there are but few cells. It is only around the larger arteries and veins near the pyramids that connective tissue is in any amount, and in this there are considerable numbers of round cells, which may increase in number in almost all pathological conditions.

There is considerable power of regeneration in the kidney, which becomes less the older the individual. In the normal kidney, not only portions of epithelial cells which are destroyed in the ordinary process of wear and tear form again from the old, but new cells are formed. It is not probable that in the adult kidney portions which are wholly destroyed can be regenerated. Certainly no evidence of such regeneration is seen around the tissue destroyed by an infarction. Hypertrophy of the remaining glomeruli can be seen in kidneys in which there has been extensive destruction, and the same thing is probably true of the tubules.

In order that the kidney may preserve its normal structure and function a number of conditions are necessary. The blood must enter the kidney in the proper manner and under the normal pressure. The circulation of the blood in the kidney must not be impaired. There must be no impediment to the venous outflow. The small veins in the kidney are numerous, the blood-pressure within them is low, and central or local circulatory disturbances easily produce passive congestion. Degenerative lesions of the epithelium are produced not only by the retained venous blood, but the direct pressure exerted on the cells by the enlarged vessels probably has some effect. Free exit must be given to the products of secretion. The ureter has thin walls; it pursues a long course, and is little protected from pressure. There is a series of pathological conditions in the urinary tract which can increase the pressure within the tubules, and this leads to alterations of structure. The character of the blood coming to the kidney must be normal. The blood may contain substances which are directly injurious to the kidney and affect its tissues in various ways.

The diseases of the kidney due to alterations in the character of the blood are the most numerous and the most serious, and their pathology is the most obscure. In a consideration of the lesions so produced it is obvious that we can divide them into classes dependent in part upon the nature of the injurious substance. These substances may be divided into those which are soluble and those which are insoluble. Insoluble substances too large to pass through the capillaries may lodge in some of the vessels of the kidney, and produce lesions in the tissue immediately around them. All of the lesions produced by insoluble substances are focal in character. Inert substances produce effects solely by cutting off the circulation of circumscribed portions of the tissue, as is shown by the infarction. Infectious substances contain or are composed of bacteria, which, by their growth, are capable of increasing their effects. It is possible to have infected emboli act both mechanically and by infecting the tissue. It is obvious that the bacteria can only enter into the kidney tissue by the bloodvessels or by the urinary passages, and it is frequently difficult to determine from the character of the lesion whether they have entered in either of these ways. If by the blood, they can easily make their way by growth into the tubules, and can be found there, or *vice versa*.

The chief difficulty in the study and classification of diseases of the kidney is found in those which are due to the action of soluble injurious substances; these soluble substances being carried to all parts of the kidney by the blood, produce diffuse lesions, which may be more marked in certain parts of the organ. It may be that the substances act in a special manner on certain histological elements of the kidney, or it may be that in certain parts of the kidney there is diminished resistance to their action due to causes for the most part unknown. Both classes of lesions, the focal and diffuse, may be found in the same kidney. In cases of severe staphylococcus infection focal lesions may be met with around emboli or thrombi of bacteria, and in addition there may be diffuse lesions which are due to the presence in the blood of soluble chemical substances which are produced by the bacteria either in some other focus or in the kidney. In certain of the infectious diseases, notably in diphtheria and typhoid fever, focal lesions in various organs, as in the liver, spleen, and lymph-glands, and to a less degree in the kidney, may be produced by the influence of soluble chemical substances produced by the disease. So that it is impossible to draw an absolutely sharp line between the etiology of the focal and the diffuse lesions of the kidney.

Bright, to whom we owe the first attempt to classify the diseases of the kidney, regarded those pathological conditions associated with albuminuria as representing three stages of the same disease, and as inflammatory in character. Rayer, in 1840, divided the lesions described by Bright into six different forms, and separated the albuminuria, due to venous hyperæmia, from the essential diseases of the kidney. He regarded the process as inflammatory in character. Reinhardt, in 1850, was the first to make a careful histological study of the lesions of the kidney, using the more improved histological methods available at that time. He considered that the different conditions found in the kidney represented different stages of one and the same disease, the small, shrunken granular kidney being the last stage in the process. Frerichs, in 1851, concluded with Reinhardt that the pathological processes in the kidney represented a single disease, and differences in the anatomical appearances were due to different stages of the process. This view as to the unity of the diffuse pathological processes of the kidney was opposed in England by Wilkes in 1850, and in Germany by Bartels in 1875. Wilkes concluded from clinical observations that the large, swollen white kidney did not pass into the small hard; that the lesions in the two conditions were absolutely different, and due to different causes. Bartels calls special attention to the differences in the clinical course of the disease marked by the large white kidney, as distinguished from the small red; dropsy being a prominent feature in the first, and usually absent in the last. Virchow's work on inflammation, in 1852, served to direct observation to the part played by the epithelial constituents, and he introduced the term parenchymatous nephritis to designate those cases in which lesions of the parenchyma are of chief importance. Baer, 1859, described the connective tissue of the kidney. He recognized its presence not only in the pyramids, but in the cortex, and introduced the term "interstitial nephritis" to describe that form of disease in which the lesions are chiefly found in the interstitial tissues. The diseases of the bloodvessels of the kidney were studied by Zeigler, 1879, who recognized a special form of disease—arteriosclerotic nephritis—due to these vascular lesions. Charcot, 1878, partly from clinical, partly from pathological anatomical observations, distinguishes three different forms of nephritis. The first is characterized clinically by a rapid course and diminution in the amount of urine, a large amount of albumin and dropsy, and anatomically by a large white kidney. The second clinically by a chronic course, increase in secretion of urine, a small amount of albumin, and anatomically by the small red, contracted kidney. The third form is the amyloid kidney. Weigert, 1879, published a celebrated article on nephritis in which he separated the diseases of the kidney into those which were solely degenerative and those which were inflammatory. In this article he denies the possibility of inflammatory lesions confined to the parenchyma or to the interstitial tissue. There may be degenerative lesions of the kidney of so slight a character that they do not cause either exudation from the vessels or proliferation of the connective tissue, but interstitial changes are always accompanied and preceded by lesions of the parenchyma. For him the whole process which was regarded as Bright's disease was a diffuse nephritis, in which he distinguished different forms.

The views of Weigert have been accepted by most of the German clinicians. It is the general habit now to speak of these diffuse renal lesions as Bright's disease, the term being used with more or less ambiguity. The term "nephritis" is also applied to the process.

For the present the classification of the diffuse lesions of the kidney must be founded on the character of the anatomical lesions. A classification on an etiological basis is the most scientific and the simplest, but we know little or nothing of the etiology of these diseases. Various forms of disease in other organs, particularly of the heart, are often found associated with them. Bacteriological investigation has shown in many cases the presence of certain organisms in the kidney. In most cases the bacteria are found in some other lesions and in the blood, and their presence in the kidney is but a part of a general septicæmia. Moreover, the same condition in the kidney may be associated with a variety of organisms, and the same organism may be associated with widely different anatomical lesions. Nor is there anything in these lesions to show that they are produced by the direct action of bacteria on the tissues. From the diffuseness of the lesions, from their affecting all parts of the kidney, it would seem evident that they must be due to injurious substances in solution in the blood. We know that chemical substances are produced by bacteria, and that their presence in the blood can produce various lesions, mainly degenerative in character, in the kidneys. Morse has shown that the continued injection of the sterile filtrate of virulent cultures of the staphylococcus aureus into the blood will produce not only degeneration of the epithelium, but hyperplasia of the interstitial tissue. Although it is probable that the chemical substances produced by the bacteria may play an important *rôle* in the production of renal disease, their importance may be overestimated. Even when we find renal disease associated with bacterial infection localized elsewhere the relation between the two may be the other way. The renal disease may be the primary condition, and the bacterial infection may be due to some alteration in the character of the blood which renders the tissues more prone to infection. Substances may be produced in the alimentary canal, or by the imperfect action of some one or other organ, which may exert an injurious action on the kidneys. When we speak of these lesions as diffuse, we do not mean that all parts of the kidney are equally affected. The study of the degenerations which occur in the course of infectious diseases and from the action of poisons has shown that in some cases the convoluted tubules are the most affected; in others the collecting tubules or the loops of Henle. All parts of the kidney are, of course, equally exposed to the injurious action of chemical substances, but all may not be equally susceptible. With slight differences in the intensity of the lesions, due to conditions which we do not understand and which may represent an increase or diminution of the local resistance, susceptible tissues will be affected. In the same way, in the more severe lesions of the kidney we find that in some the glomeruli are principally affected; and although these lesions in the glomeruli may be accompanied by lesions chiefly degenerative in the tubular epithelium, these may be, in part or wholly, secondary to the lesions in the glomeruli. In other cases, again, there are lesions in the connective tissue consisting in active proliferation of the cells contained in this. This acute hyperplasia of the connective tissue, although accompanied by degenerative changes of the epithelium, cannot be considered as primary to such changes. No anatomical classification can be so sharp and definite as one based on etiology. In the anatomical classification there often occur mixed forms. One form may have another grafted on itself, and an acute glomerular affection may be added to a chronic arteriosclerotic change, or to a hydronephrosis. The anatomical classification must be based on the character of the lesions as determined by the histological examination. Although the microscopic lesions are accompanied by more or less definite alteration in the gross appearance of the kidney, yet the connection is not sufficiently definite to enable us to determine by these gross appearances the one form of lesion from the other. From my study of these diffuse lesions of the kidney it has seemed to me that the following classification might be adopted. I include here both the acute and chronic, although this paper has reference only to the more acute lesions. It must be understood that in using the word nephritis we do not necessarily imply that the lesions fall under the rubric of inflammation, but the word is used as signifying simply disease of the kidney. It might possibly be well to substitute for the word nephritis the term "nephropathy," although the objection to this is a complication of an already complicated nomenclature.

I have adopted the following as perhaps the most suitable classification of diffuse nephritis which we can make from an anatomical basis:

ACUTE DIFFUSE NEPHRITIS. (*a*) *Acute degenerative nephritis.* In this are included degenerative lesions of the epithelium, embracing cloudy swelling, hyaline, fatty, and dropsical degeneration, and often complete necrosis, without lesions other than degenerative, in the glomeruli or in the interstitial tissue. This occurs chiefly in infectious diseases, in jaundice, in anæmia, and as the result of the action of certain poisons. The kidney is slightly swollen or unchanged in size, rather paler and more opaque on section; the markings may be obscure or more prominent than normal. There is often albuminous exudation in the glomerular capsules and in the tubules.

(*b*) *Acute glomerular nephritis.* The essential changes consist in acute lesions in the glomeruli. There may be acute proliferation of the endothelium of the vascular tufts, hyaline and fibrinous thrombi in the vessels, accumulation of leucocytes in the vessels, degeneration of the vessel-wall, etc. These changes in the vascular tufts of the glomerulus can take place with or without changes in the capsular epithelium. The changes in the capsular epithelium consist in degeneration and proliferation. The capsular space may contain an albuminous hemorrhagic or fibrinous exudation. The changes in the vascular tufts and in the capsule are so frequently combined in various degrees that they cannot be separated into two sub-classes. The glomerular lesions are accompanied by degeneration of the tubular epithelium, necrosis, and exfoliation. Often there are dilatation of the tubules and œdema and cellular proliferation of the intertubular tissue. There may be more or less hemorrhage in the tubules.

This affection occurs in infectious diseases, notably in acute endocarditis, measles, and diphtheria, or as an independent affection. The kidney is usually increased in size. The capsule easily strips off; the surface is pinkish and mottled with points of ecchymosis. On section the cortex is wide, rather paler and more opaque, markings obscure; glomeruli pale, enlarged, and prominent. Pyramids often congested. The tissue moist and pits on pressure. While these appearances are usually marked, lesions of the glomeruli may be found with but little macroscopic change in the kidney.

(*c*) *Acute hemorrhagic nephritis.* The essential change consists in hemorrhage in the tissue combined with degeneration of the epithelium. The hemorrhage is chiefly found in the capsule of the glomeruli and in the tubules. The degenerative lesions may be extensive and lead to necrosis and exfoliation. Œdema, hemorrhage, and cellular infiltration are often found in the intertubular tissue. The kidney is enlarged, hemorrhages are found in the capsule; the surface is dark red, with numerous ecchymoses. On section the cortex is swollen and sprinkled with dots and streaks of ecchymosis. Only three cases of this were found in my series.

(*d*) *Acute interstitial non-suppurative nephritis.* The essential lesion consists in acute proliferation of the cells in the intertubular tissue. The proliferation takes place mainly from the vascular endothelium. The cells lie within and without the vessels. They are large and similar to the endothelial cells of young granulation-tissue. They are found chiefly in the intermediate zone of the kidney between the pyramids and the cortex. In the cortex they are both generally diffused and in areas chiefly around the glomeruli. There is more or less degeneration and necrosis of the tubules, affecting chiefly those in the areas of cellular infiltration. Leucocytes in small numbers may be found in the intertubular tissue among the other cells, in the degenerated epithelium, and in the lumen of the tubules. The glomeruli are not affected. This affection occurs in acute infectious diseases, notably in diphtheria and scarlet fever. The kidney is large, pale, somewhat mottled; on section moist, opaque, markings obscure, and milky fluid can be pressed from it.

SUBACUTE GLOMERULAR NEPHRITIS. The essential lesions are in the glomeruli. They consist in swelling and nuclear increase in the vascular tufts and obliteration of the vessels by hyaline degeneration, both of the cells and the vascular walls. These changes in the tufts are often combined with proliferation and desquamation of the capsular epithelium with connective-tissue ingrowth. There are extensive degeneration, necrosis, and desquamation of the tubular epithelium. The intertubular tissue is the seat of œdema and connective-tissue formation. There is often a previous history of exposure to cold or of an infectious disease, and it may occur after rheumatism or endocarditis. The kidney is enlarged, the capsule may cling slightly to the surface; the surface is pale or slightly mottled. On section cortex increased in width, pale, opaque, markings obscure, glomeruli pale, consistency increased. There are gradual transitions between this and the acute and chronic glomerular nephritis.

CHRONIC DIFFUSE NEPHRITIS. (*a*) *Chronic glomerular nephritis.* The essential lesions are in the glomeruli. They consist in extensive hyaline degeneration of tufts and of entire glomeruli, and obliteration of capillaries. Every transition may be seen between these glomerular lesions and those in the subacute form. There may be some increase in the capsular epithelium and connective-tissue formation within the capsule. The tubular epithelium shows extensive degeneration and destruction. Entire tubules are destroyed, often being represented by the thickened irregular membrana propria. There is a general and diffuse increase of the connective tissue affecting almost equally all parts of the kidney. This condition is usually found as an independent affection or may be combined with acute infections of various sorts. There is often a history which points to a previous acute or subacute affection. The kidney may be slightly larger than normal, of normal size, or considerably smaller. The capsule is often adherent, the surface even, not granular, pale. On section the cortex varies in width; it may be quite small. It is opaque, whitish, the markings obscure; the glomeruli not visible or pale, and the consistency of the tissue is greatly increased.

(*b*) *Chronic arteriosclerotic nephritis.* The essential lesions are in the arteries and consist in those changes which are known as arteriosclerosis. There is degeneration of the epithelium of the tubules with more or less complete destruction. The degeneration takes place slowly, and at any given time sections may show only a slight degree. Atrophic changes in the epithelium are common. The lesions may affect almost equally all parts of the kidney, or appear in areas corresponding to the distribution of those arteries which are most affected. There is a general increase in the connective tissue, though large areas of tubules may be found with no increased tissue between them. The lesions are never so diffuse as in the glomerular form. This condition of the kidney is found accompanying a general arteriosclerosis affecting all the arteries of the body, or the lesions may be most marked in the renal arteries. The kidney varies in size; it may be slightly larger, or of normal size, but it is usually very much smaller. The capsule may or may not be adherent.

The surface is more or less irregular and granular. The color red, often cyanotic. On section the cortex may be much diminished in size, of a dark-red color, the markings obscure, the glomeruli injected; the arteries in the intermediate portion are evident and often project above the cut surface. The pyramids show venous congestion. The consistency of the kidney is greatly increased. The lesions are due to a combination of causes: to the diminution in the blood-supply, to the impairment of the function of the vessels by the destruction of the muscular coat, and to the accompanying venous congestion. Most of the cases of contracted kidney belong to this class.

(*c*) *Chronic degenerative and interstitial nephritis.* To this class belong those cases of contracted granular kidney which occur without primary arterial lesions. It is difficult to give a name to the condition, for there is no single change which predominates. Degeneration, atrophy, and destruction of the epithelium in various places are found. There is a general increase in the connective tissue more diffuse than in the arteriosclerotic nephritis, but not so diffuse as in the chronic glomerular form. The increase in the connective tissue is most intense where the degeneration of the epithelium is most marked. Lines of connective tissue extend to the surface, and by their contraction produce depressions. Very minor degrees of change, which may consist in small areas of cellular infiltration, with hyperplasia of the connective tissue extending down from the capsule, are very commonly found. The essential lesion seems to be a slow degeneration of the epithelium, followed by connective-tissue hyperplasia. The macroscopic appearances of the kidney vary extremely, following the different degrees of the lesion. The gross and microscopic conditions may be complicated by lesions of another character.

SENILE NEPHRITIS. In the senile kidney the chief lesions are those due to disease of the vessels. These vascular lesions are accompanied by impairment in the power of regeneration. Previous lesions of the kidney, even though slight in character, may gradually make their influence felt in impairing the resistance of the tissue. The epithelial lesions may consist chiefly in atrophy. Microscopically the kidney is usually more or less injected and atrophied. On microscopic examination the epithelium of the tubules is degenerated, small, and atrophic. The formation of yellow pigment in the atrophic epithelium is frequently seen. There is some general increase in the connective tissue, but this is chiefly marked close beneath the capsule, and may extend from here in lines into the cortex.

AMYLOID NEPHRITIS. In the amyloid nephritis, as the name implies, the characteristic lesion is the amyloid infiltration of the blood-vessels. This may occur alone and may produce certain lesions of the tissue, or it may accompany any of the other forms of nephritis. When amyloid infiltration affects the glomeruli alone lesions closely resembling those in other forms of glomerular nephritis may be produced.

In this paper I shall give the results of the study of fifty-two cases of nephritis. They embrace acute, subacute, and chronic glomerulonephritis, acute interstitial and acute hemorrhagic nephritis. The larger part of these kidneys come from autopsies made at the Boston City Hospital, in which the bacteriological cultures of all organs were made. A part were brought to the pathological department of the Harvard Medical School for diagnosis, only the kidneys being examined; and in part they consist of old specimens in which little or nothing was known of the accompanying disease and no bacteriological examination made. The clinical histories of most of these cases are unsatisfactory. Many of them have gone into the hospital in a moribund condition, and nothing could be learned concerning them. In others little attention was paid to the examination of the urine, as attention clinically was not prominently directed to the condition of the kidneys.

The methods used for histological examination consisted in making frozen sections, in hardening the tissues in various ways, and examining sections of the hardened tissues after imbedding in celloidin and paraffin. All the modern methods of staining were used. A great deal of importance should be attached to the study of frozen sections, because only in this way can the extent of the degenerative lesions of the epithelium be fully recognized. This method was found far more satisfactory for this purpose than by the use of osmic acid.

ACUTE GLOMERULO-NEPHRITIS. The glomerular lesions in diseases of the kidney were recognized by the early investigators, but it is only of late that the important part which they play has been appreciated. Virchow described the increase in the number of cells in the glomerulus, and gives a figure illustrating the condition. Förster has described the increase in cells in the capillaries, thickening of the capillary wall, and proliferation and desquamation of the epithelial cells of the capsule. Klebs was the first to study these changes of the glomerulus more closely. He describes in the kidney of scarlet fever slow proliferation and degeneration of the capsular epithelium. He also describes an increase of the cells in the glomerulus, which he thinks comes from a proliferation of the cells of the connective tissue between the vessels. Langhans, in two publications in Virchow's *Archiv*, describes the lesions in the capillaries. He thinks that these lesions consist in proliferation of the capillary endothelium, leading to the accumulation of cells within the capillaries, and the formation of a protoplasmic network which completely occludes the vessels. Hausemann describes the cells in the glomerulus as lying between the capillaries and due to proliferation of the connective tissue.

Ribbert speaks of the importance of glomerular lesions in all cases of nephritis. He found them not only in acute scarlatinal nephritis, but in subacute and chronic cases. Welch describes the glomerular lesions observed in the human kidney as well as those produced experimentally in white rats by the injection of cantharidin. He proposes the term desquamative glomerulitis to describe those cases characterized by proliferation of the capsular epithelium with accumulation of cells in the capsules, and the intracapillary glomerulitis to describe those cases in which accumulation of cells inside of the capillaries is the chief lesion.

I have observed twenty-eight cases in which lesions of various degrees of acuteness were found in the glomeruli, and in which, although lesions in other parts of the kidney were found, the glomerular lesions from their intensity appeared to be primary. In placing them among the acute cases the character of the lesions, and not the clinical history, has been considered. In those cases in which a clinical history referable to the kidneys has been obtained the lesions which have been considered anatomically as acute have agreed with the clinical history. In determining the acuteness of the process from an anatomical point of view the abundance of cells and the slight character or absence of connective-tissue increase have been mainly considered. Although these glomerular lesions vary, the pathological processes found in the glomeruli are too often combined to warrant us arranging them into different classes.

I shall take up separately the different processes found in the glomeruli. Proliferation of the cells in and among vessels of the glomeruli was observed in all but two cases. In a number this was the only change observed. On microscopic examination with low power the glomeruli are prominent from their increased size and the abundance of cells. They fill completely the capsular space and the capillary loops adjacent to the tubule, which causes the glomerulus often to project a considerable distance into the tubule. On section these masses projecting into the tubules may be cut across, appearing then as portions of capillaries lying within the tubules. Serial sections and sections cutting the glomerulus in other directions will show that they are always connected with the mass of capillaries. In most of these glomeruli there is no appearance of division of the capillaries into separate masses. In those cases regarded as most acute the capillary walls could be clearly distinguished. The walls of the capillaries are thickened and refractive. The cells in some cases can be seen to lie within the capillaries, but this is only the case in cross-sections of the vessels and in very favorable longitudinal sections. The vessels are so small, and their course so tortuous, that only by the most exact focussing can we distinguish those lying in the vessels from those outside. The special cells in the vessels are not leucocytes. In many cases leucocytes are found among them, and in some cases they are very abundant. The polynuclear leucocytes can always be distinguished from the other cells within the capillaries by the form and characteristic stain of the nucleus. The other cells also differ from lymphoid cells. The nuclei are granular, and they stain brightly, but not so brightly as the nuclei of leucocytes. In some cases the protoplasm of the single cells is difficult to make out, and masses of protoplasm appear to be joined together, forming an irregular protoplasmic network within the capillary similar to that described by Langhans. The cells can be distinguished from the nuclei of the epithelium by their oval shape and the more vesicular character of the epithelial nuclei. In some cases very large cells were abundant, and granular protoplasmic masses were found within the capillaries. Many of these intracapillary cells are similar to those found in acute interstitial nephritis, which can be clearly shown to lie within the vessels, and to be probably derived from proliferation of the endothelium. In all of these cases the absence of red blood-corpuscles in the capillaries of the glomeruli is conspicuous.

In specimens hardened in Flemming's solution extensive fatty degeneration was often found in these cells, both those without and those inside the capillaries. In the specimens which were hardened in corrosive sublimate, or in fluids into whose composition this entered, the red corpuscles were stained vividly, and in some cases none, in others very few were found within the vessels. With the increase of cells within the vessels there was found associated a thickening of the wall which seemed to take place on the inside. The edge toward the lumen becomes irregular, and in some cases the lumen is almost entirely obliterated. These thickened vessels give a distinct hyaline stain, and seem to represent the beginning of the more marked degeneration of the glomerulus described in the subacute and chronic forms.

In addition to the cells a more or less irregular, somewhat granular material was found in the capillaries. It sometimes forms a reticulum among the cells, or by itself. This material did not stain as fibrin, and may have been composed of the remains of degenerated and necrotic cells. In some cases small hyaline masses were found in this reticulum.

The extent to which the leucocytes take part is interesting in view of the fact that some observers have considered most of the cells within the glomerulus to be leucocytes. In but one case were they found in the glomerular vessels in any abundance. Some of the capillaries in this case were filled with them, and they were found in considerable numbers in the glomerular spaces and in the tubules. This is interesting in view of the fact that emigration of leucocytes through the glomerular vessels has been denied. In some sections in this case they were seen in the act of emigration, part of the cells being within and part without the vessels, and both those in the glomerular capillary and those in the tubules were most probably due to emigration from the glomerular vessels. In those cases in which necrosis of groups of vessels was seen leucocytes and nuclear detritus were found in the necrotic tissue. Hyaline thrombi were found in the vessels in three cases. In one of these all of the capillaries and some of the glomeruli were occluded; the thrombi were perfectly homogeneous, highly refractive, and gave the characteristic fibrin-reaction with Weigert's fibrin-stain. Ribert describes such hyaline thrombi in the kidneys of rabbits after staphylococcus infection, and they have been found in a variety of other acute infections. They appear to be more common in animals than in man. In one case they were found in a child with staphylococcus septicæmia, and in the other two cases no cultures were made. Fibrin within the vessels and the capillary spaces, and in the tubules, was found, both associated with hyaline thrombi in the glomeruli and without them. In one case so much fibrin was present that it must have played an important part in the formation of casts. Necrosis of glomeruli affecting small masses of capillaries was found well marked in two cases, and a slight degree was seen in some of the other cases. In the necrotic portion the details of the tissue could not be made out. The whole tissue was converted into an indefinite granular mass in which Weigert's stain showed some filaments of fibrin. Leucocytes and nuclear detritus were found in it. This definite necrosis of the tissue of the glomerulus differs from the formation of hyaline. The hyaline tissue appears both as hyaline thickening of the capillary walls and as the formation of hyaline drops in the protoplasm of the cells within the capillaries. Proliferation of the capsular epithelium plays a prominent part in the glomerular changes. In some cases this was so marked that on superficial examination the other lesions could have been overlooked. Although more marked in some cases than in others, it is impossible to divide the cases according to this lesion. It was especially evident in two cases, but was present to some extent in nearly all. Even in the most typical of the intracapillary cases among the cells there were some which, from their large size and the vesicular character of the nuclei, must have been derived from the epithelium. The proliferating capsular epithelial cells formed crescentic-shaped masses between the glomerular tuft and the capsule. The cells were usually flattened by pressure and the nuclei deformed. In some the nuclei had lost their power of staining, and the cells were converted into a more or less granular, apparently necrotic mass. Hypertrophied capsular cells connected with the capillaries by a long footstalk were occasionally found. Nuclear figures in small numbers were found in the capsular epithelium. Dilatation of the tubules was often observed, and it is difficult to explain. It is possible that it may have been due in part to pressure exerted on the tubules in some parts of their course by the œdematous intertubular tissue. In part it may be explained by a blocking up of the tubules by casts and epithelial detritus. Fatty degeneration of the epithelium was found to some degree in almost all cases, but was in none excessive. The extreme degree of fatty degeneration which is found in phosphorus-poisoning, jaundice, and sometimes in acute pneumonia, was never present. The fatty degeneration was generally diffuse, and the fat was in small granules. Granular and hyaline degeneration of the epithelium was found both with and without the fatty degeneration. In the hyaline degeneration single cells were found enlarged and filled with large and small hyaline drops. Sections of single tubules were found filled with hyaline material. The nuclei of such cells were in some cases fairly well preserved, in others they either did not stain or were small and solidly stained. Single hyaline degenerated cells or masses of hyaline cells partly preserving the globular form, and partly moulded into hyaline cylinders, were often found in the tubules, evidently the result of desquamation of the epithelium higher up. In some of the dilated tubules the epithelium was simply atrophic. Necrosis affecting single cells and groups of cells was frequently seen. The single necrotic cells could be distinguished from the others by the homogeneous staining of the protoplasm and the changes of the nucleus. In some cases the epithelium was enormously swollen, very clear, and filled with small spaces. A very common condition found in the tubules was the presence of masses of small circles which filled up the entire tubule, the edges of the circles forming a fine reticulum. Granules were often present in the edges of the circles, both within and around them. With fuchsin the circles and granules often stained as brightly as do the nuclei. A reticulum of this character may be seen in the simple degenerative lesions of the kidney; it was often found in the capsule of the glomerulus. In one case it was very prominent, almost every tubule being filled with it. I do not know the source and the nature of this peculiar material, which may be met with in other places. I have never seen any appearances which make me believe that it can be a substance secreted by the cells. The swollen ends of the cells may be so cut by the microtome knife that they appear as circles in the tubule; but it is obvious that such an explanation will not suffice. An albuminous secretion coagulated by the hardening-agents does not take such a form, but is represented by a granular or homogeneous mass, this depending on the hardening-agent. The most marked change in the interstitial tissue of the kidney is œdema, and the increased size of the kidneys, in one case reaching 880 grammes, is generally due to this. The space between the tubules may be very wide, and is filled with a thin, sparse reticulum, in the midst of which run the vessels. The œdematous condition of the kidney is also evident macroscopically. The tissue is more succulent than normal, and in some cases pits on pressure. In the acute cases there is no actual increase in the connective-tissue fibrillæ. In those cases in which the lesions of the glomeruli approached the subacute form, as is shown by the beginning lobulation and hyaline degeneration of groups of vessels of glomeruli, the interstitial tissue is denser and contains more cells. The cellular increase takes several forms. In the intermediate zone there is found small cell-infiltration around the larger vessels, particularly around the veins. These cells are small and similar in character to the lymphoid cells of lymphatic tissue and the blood.

As we have said, in the normal kidney small collections of such cells, probably representing small areas of lymphatic tissue, are often found around these vessels, and the increased number of cells may represent hyperplasia of this tissue. In other cases the cells have a different character. They are large; the nuclei stain brightly and contain considerable chromatin and reticulum. The protoplasm is finely granular and stains more intensely than the protoplasm of the epithelial cells. These cells are frequently found within the vessels, and many of them certainly come from proliferation of the endothelium. In several cases these cells were present in variable numbers, and in one case they were so numerous as almost to warrant us in regarding this case as a mixed form of glomerular and interstitial nephritis. In one case the connective tissue in the vessels showed the lesions of arteriosclerotic nephritis, and superadded to this there were acute lesions of the glomeruli.

In all cases the kidneys were enlarged. In adults the weights varied from 400 to 880 grammes; average weight 517 grammes. Orth gives the normal weight of adult kidneys in man as 320 grammes, and in women as 293 grammes, which is slightly under the average I have found. The weights were taken after removal of the capsule and adherent tissue of the pelvis. The capsule is usually hyperæmic; it strips off easily, even parting itself from the swollen kidney after section. The surface is smooth, or it may be irregular from accompanying infarctions or previous disease. The color varies, being usually of a light pinkish tinge, and mottled from varying degrees of congestion. There are usually small whitish foci varying in size, marking areas of more intense fatty degeneration and small points of ecchymosis. On section the tissue is distinctly moister than normal. A thin, opaque fluid is easily squeezed from it, and it pits on pressure. The cortex is increased in size, and the markings are obscure. On section of the cortex the small opacities and ecchymoses may be observed as on the surface. The glomeruli are pale and bloodless. In most cases they are easily visible and stand out from the cut surface, appearing as grains of fine sand sprinkled over it, and are rather more refractive than normally. The pyramids are usually enlarged, but relatively much less than the cortex, and may be hyperæmic.

The presence of bacteria in the kidneys or in the accompanying acute lesions was noted in eighteen cases. Their presence was determined by cultures, by microscopic examination of cover-slips and in sections. In some cases inoculations of animals with pure cultures of the bacteria found were made. No anaërobic cultures were made. Little or no attention was paid to the colon bacilli, and in only one case in which they were found alone is their presence mentioned. In the routine cultures made at autopsy they are found so commonly and in such different conditions with and without lesions, and accompanying bacteria which may be regarded as the cause of the lesion, that we are disposed to consider them as having no specific and probably no pathogenic properties. They probably enter the tissues from the alimentary canal immediately after death or under conditions when the resistance of the tissues to their growth is diminished. There has been a notable diminution in the finding of colon bacilli at autopsies since the use of the refrigerator system, by which the bodies are kept at a temperature of 32° in the Morgue of the Boston City Hospital. Usually the bacteria found in the lesions in the kidneys were simply part of a general septicæmia. The organism most commonly found was the pneumococcus. This was found in eleven cases; in three streptococci were found; in one staphylococci; colon bacilli in one without any accompanying organism, and in two the cultures were sterile. Of the sterile cases, the kidney-lesions in one followed acute articular rheumatism, and in the other there were no lesions elsewhere. There was some general agreement between the character of the lesions and the sort of bacteria present. The most typical cases of the intracapillary glomerular lesions accompanied infection by the pneumococcus. In the streptococcus infections leucocytes entered more prominently into the process, and there was more of the acute interstitial change. In the single staphylococcus infection the hyaline thrombi in the glomeruli were the most prominent accompanying acute lesions. Endocarditis was present in ten cases. Nine of these were due to pneumococci, and one to streptococci. The endocarditis was acute and usually of the ulcerative form. In most cases the lesion was primary, and in a few only represented an acute form engrafted on an old lesion. There is a well-marked relation between pneumococcus endocarditis and that form of glomerulo-nephritis in which the intracapillary lesions are most prominent. In one case of general peritonitis, due to pneumococcus, from an autopsy of Dr. Wright's at the Massachusetts General Hospital, the kidney lesions were of the same type as those in acute pneumococcus endocarditis. In my earlier investigations so many cases of acute endocarditis were found with intracapillary lesions that I was disposed to consider pneumococcus endocarditis as the single associated condition. The pneumococcus has been present in the form of a general septicæmia in practically all cases in which the acute renal lesions were found.

In the Boston City Hospital acute croupous pneumonia, due to the pneumococcus, forms a large portion of the autopsy material. On recently reviewing the cultures made in acute croupous pneumonia we have found that out of 110 cases 107 gave pneumococci on culture. Glomerulo-nephritis has been found to accompany the pneumonia in but one case, and in this, both from the character of the lesions in the kidney and from the clinical history, it was evident that the kidney lesion was primary. Interlobular pneumonia, with abscess due to staphylococci, was found in one case. It is very remarkable that the pneumococcus endocarditis should be so frequently accompanied by nephritis, and the acute croupous pneumonia, due to the same organism, so rarely. Erysipelas, with streptococcus infection, was found in one case; measles in two; syphilis, with the constantly accompanying streptococcus infection, in two, one of which was combined with measles. Acute articular rheumatism was found in one case; acute streptococcus infection following abortion in one; scarlet fever in one; diphtheria in one; gonorrhœa in one, and in one acute pneumococcus peritonitis. The presence or absence of œdema was noted in fourteen cases. Of these it was present in ten cases. In seven of these cases there was acute endocarditis, with extensive valvular lesions, in one erysipelas, and in one acute articular rheumatism.

SUBACUTE AND CHRONIC GLOMERULO-NEPHRITIS. I have included with the acute cases the cases of subacute and chronic glomerulo-nephritis because their consideration obviously follows in this place, and every gradation from the acute to the subacute and chronic can be followed.

This condition of the kidney has been known under various names. It is the large white kidney of Wilke and the chronic parenchymatous nephritis of modern writers. There is no doubt that the amyloid kidney was and still is frequently confounded with it, and without staining for amyloid they often cannot be distinguished. In the amyloid kidney there is the same degeneration of the epithelium, due most probably to the same cause. Disease of the glomerulus interferes with the circulation within it. In the amyloid kidney the vessels of the glomeruli are occluded by amyloid material deposited in the walls of the vessels and between them. In the chronic glomerulo-nephritis the occlusion is brought about by hyaline degeneration. In both the duration of the process varies, and in both there may be extensive destruction of the parenchyma, followed by a general increase in the connective tissue. It is convenient to separate them by the presence of amyloid in one; but apart from the amyloid the process is very similar, although the deposit of amyloid in the glomerulus is not preceded or accompanied by cellular proliferation. In a review of the lesions of these cases the glomeruli claim our chief attention. Much that we have stated of the condition of the glomeruli in the acute glomerulo-nephritis is clearly applicable here. Slight differences in the degree of involvement may be found in different glomeruli and in different parts of the same glomerulus. All of them show an increase in the number of cells. In places conditions resembling the acute forms are seen, the cells being greatly increased in number, and lying for the most part within the capillaries. A condition of lobulation of the glomerulus is one of the most prominent lesions. Even in the acute form a division of the glomerulus into more or less definite lobules is often seen. In the more marked form the glomerulus appears to be divided into a number of definite lobules, the separation between them extending down to the root. Cross-sections of the glomerulus and root usually show four to six such lobules. In sections passing through the glomerulus in the other direction as many as eight distinct and separate masses can be found; with the lobulation the hyaline degeneration of the glomerulus advances. The hyaline degeneration appears first in the outlying capillary loops, those most distant from the root. At first only small portions become hyaline, and by gradual extension whole lobules and even whole glomeruli are so altered. The hyaline material is homogeneous, it stains brightly with eosin and with acid-fuchsin, and in alcoholic specimens saffranin gives it a faint bluish or lilac hue. The hyaline appears to be formed in two ways, finally becoming united into a single mass. The membrana propria of the capillary wall becomes swollen, and takes on a hyaline stain. Cells lying within and between the capillaries show hyaline droplets in their interior just as do the cells of the tubules, and the hyaline material so formed may become fused together with the production of irregular hyaline masses in which remains of nuclei and particles of chromatin may be occasionally found. Such hyaline material, unlike the hyaline thrombi, does not take the fibrin-stain. In the hyaline masses small spaces, with here and there red corpuscles or leucocytes, mark the vessels. Dilated vessels filled with red blood-corpuscles are occasionally found. At first there seems to be no increase in the number of nuclei. They are crowded together in a hyaline mass and show various differences in form and staining. Most of them are irregular and elongated, and stain homogeneously. The protoplasm of the cells belonging to them is merged into the hyaline mass. Very thin sections in places where the hyaline formation is not advanced enable us to gain some idea of its mode of formation. Occasionally capillaries are found cut longitudinally in which the lumen is exceedingly small and irregular, and the wall thickened apparently by the formation of hyaline on the inner wall of the vessel. Nuclear detritus is found to some extent in the hyaline material. All the cases in which fresh examinations were made, or in which portions of the tissue were hardened in Flemming's solution, showed a considerable fatty degeneration of the cells of the glomeruli. The capsular epithelium in most cases shows some hyperplasia. Flattened epithelial cells derived from the capsule are found in the capsular spaces and between the lobules of the glomerulus. In some cases the growth is excessive, and sections passing at right-angles to the root of the glomerulus showed the single groups of hyaline vessels completely surrounded by epithelial masses derived from the capsular epithelium. The nuclei in the epithelial cells are frequently irregular in form and atrophic, and the cells converted into a more or less granular mass. Various degrees of connective-tissue formation take part in the capsular change. Bands of connective tissue extend from the capsule into the epithelial masses, more or less dividing them, and connective-tissue fibres are shown by the Van Giesan stain among the cells. There is no evidence of connective-tissue formation from the capsular cells, but in all instances it seems to have gone into the masses from the connective tissue of the capsule. The connective tissue of the capsule is found thickened and the cells in it increased in number. The hyaline masses of the glomerulus are often found adherent to the capsule, and in one specimen there was a long, projecting mass of connective tissue which extended from the capsule down to the root of the glomerulus, and which contained several wide vessels. The epithelium of the tubules showed extensive degeneration which did not differ materially in kind from that described in the acute forms. The tubules are found dilated and filled with desquamated cells, hyaline material, and glomerular débris. In one case the epithelium of the dilated convoluted tubules was atrophic and the cells crescentic in form. The tubules in this case contained great numbers of these crescentic-shaped cells, which seem in part to be due to desquamation in the tubules; in part to come from the glomerular capsules in which there is extensive hyperplasia. Hyaline degeneration of the epithelium was found in every case. There is a varying degree of involvement of the connective tissue of the kidney. It is always slightly increased, and in the more chronic cases there is a general growth extending diffusely throughout the tissue. The diffuseness of this connective-tissue formation explains the absence of granular contraction of the surface, even though the kidney, as a whole, may be atrophied. That the subacute and chronic glomerulo-nephritis represents a more marked condition of lesions of the same general character as those found in the acute form is evident. In both the glomerular changes are the most striking lesions. Changes similar to the acute, consisting in the increase of cells in the capillaries and hyperplasia of the capsular epithelium, are found in the chronic. In the chronic there is a greater degree of hyaline degeneration, partly from conversion of the cells into hyaline, partly from hyaline degeneration of the walls of the vessels, followed by contraction, by which the lobulation becomes pronounced. The degree of the connective-tissue formation seems proportionate to the degree of epithelial degeneration and desquamation, and probably follows it. Macroscopic appearances of these kidneys vary greatly; they were usually of somewhat increased weight. The lowest weight found in the six cases at the Boston City Hospital was 300 grammes, and the highest 425 grammes.

Of the two cases in which the lowest weights were found, one was a boy of twelve years, and the other a girl of sixteen years. The average weight of the six cases was 395 grammes. The macroscopic appearance of the kidneys also varies. The capsule strips off easily. The cortex of the kidney is smooth, rather paler than normal, and the surface is marked with small, white, opaque spots, and by small foci of ecchymosis. On section the markings of the cortex are obscure, the consistency of the kidney is increased. The glomeruli may be seen on the cut surface as small, pale, projecting points. No complete history of the duration of the disease could be obtained. The longest history was that of six months, and the shortest two months. An accompanying acute rheumatism was found in two cases; two attributed the disease to having taken cold, and in one case there was an acute endocarditis engrafted on an old lesion. Nothing of any importance was learned from the bacteriological examination.

ACUTE INTERSTITIAL NEPHRITIS. This form of nephritis was first described by Wagner under the name of lymphomatous nephritis, and he makes it one of the four forms of Bright's disease. He says it is most frequently found in scarlet fever, but may be found in typhoid. The kidneys are large and pale. Hemorrhage may be present in slight degree or entirely absent. The most characteristic lesion is an accumulation of round cells of various sizes, which are most numerous around the glomeruli. Others have described these lesions in the interstitial tissue, but they have not been regarded as sufficiently marked to constitute an anatomical type of nephritis.

Orth has an interesting description of the process in his text-book of *Pathology*. He says he has seen kidneys in which there were none or very very slight lesions of the epithelium and glomeruli, but in which there was a small cell-infiltration in the interstitial tissue. He describes such kidneys as large and pale. Regarding the origin of the cells, he says they have a large, round, vesicular nucleus, and differ from the ordinary emigrating leucocytes. He thinks they may be derived from proliferation of the tissue-cells, although their position around the veins would show that emigration also plays a part in their production.

There is a liability to confusion in giving the name acute interstitial nephritis to this process because this name is frequently used to describe purulent nephritis. The lesions in the interstitial tissue are so marked that no other name is fitted to describe them. The essential lesions are in the interstitial tissue, and are so prominent and accompanied by so little change in the parenchyma that they cannot be regarded as secondary to parenchymatous lesions. While there is no doubt that lesions of the parenchyma of a tissue, when they are sufficiently extensive and when long enough time elapses, will be followed by a growth of interstitial tissue which has the effect of making good the loss, there is equally no doubt that there can be primary lesions of the interstitial tissue. The cells in this case are in a way similar to those seen in acute hyperplasia of the connective-tissue cells, particularly those of the walls of the bloodvessels occurring under other conditions. Similar cells to these may be found in rapid proliferation of subcutaneous tissue in the vicinity of wounds, and it is probable that they have the same origin. The cells seem in large part to come from the endothelium of the bloodvessels, the small veins, and capillaries. So far as I have been able to see, the arterial endothelium takes no part in the process. The cells are found in the greatest numbers in the lower portion of the cortex, which we have designated as the intermediate zone, though by no means confined to this. There are both a general and a focal increase in the cells. The focal increase is found chiefly around the glomeruli, extending from these into the surrounding intertubular tissue. The cells of the connective-tissue capsule of the glomeruli and the capsule around this take part in the process. Among these cells leucocytes are present in variable numbers, and the process is probably accompanied by emigration. The leucocytes are found both in the interstitial tissue, between the cells, and in the degenerative and necrotic epithelium of the tubules in their midst, and in the lumens of these tubules. None of the large cells are ever found within the tubules. In all of the cases there is more or less degeneration of the epithelium, which is more marked where the interstitial cellular infiltration is most prominent. Elsewhere throughout the kidney the degeneration, though present, is not so marked. In those cases in which fresh examinations were made fatty degeneration was found. In none of the cases were the glomeruli affected. The degeneration of the tubular epithelium in the interstitial infiltration may be partly due to pressure exerted by the masses of cells. It is probable that in this case we must assume the presence of a toxic material which exerts its action on the constituents of the kidney tissue, and which at the same time leads to a degeneration of the parenchyma and proliferation of the interstitial tissue. The macroscopic appearance of the kidneys is characteristic. They are enlarged ; the capsule is often so tightly stretched that on section it bursts from the tissue of the kidney. The increase in size of the kidneys is in the cortex. Beneath the capsule the kidneys are pale, more opaque than normal, and with areas of hyperæmia and few ecchymoses. On section they often present the appearance of abscess-formation. Just above the pyramids extending in lines up into the cortex there are opaque masses appearing as abscesses from which a purulent-looking fluid can be squeezed. The markings of the cortex are completely obscured ; the glomeruli are not visible.

In the nine cases of this which I have seen, in one the age could not be ascertained ; one was from a woman aged thirty years, and the others were from children, the oldest eleven years.

Bacteriological examination was made in but five cases, and in all of these general streptococcus infection was found, combined in one case with a general infection with the diphtheria bacillus. The associated disease was ascertained in eight cases. In four of these there was scarlet fever; in one diphtheria and scarlet fever; in two diphtheria, and in one endocarditis following abortion. In none of the cases was there any œdema, and in none was attention particularly directed to the kidneys during life.

Acute Hemorrhagic Nephritis. In this the chief lesions consist in hemorrhage accompanied by a varying degree of degeneration of the epithelium. Only three cases were met with. Two of these were from typhoid fever, and one from a case of acute endocarditis with infectious pneumonia. The character of the pneumonia was not described, but it was probably embolic. The hemorrhages in these cases were added to acute degeneration. In most of the cases the hemorrhage appears to take place from the glomerulus. In one case there was extensive hemorrhage in the tubules without any change in the glomeruli.

TUBERCULOUS OCCLUSION OF THE ŒSOPHAGUS, WITH PARTIAL CANCEROUS INFILTRATION.

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On December 3, 1895, was admitted to the University Hospital a Russian cigarmaker, aged forty-two years, who had been seven years in America.

His family history had no bearing on the case, and was entirely good. His father died at the age of sixty-eight, of pneumonia. His mother was killed in an accident, and his brothers and sisters, five in number, were living and well. There was no history of consumption or cancer in near relatives.

His personal history up to March, 1895, was good, and he could remember no serious illness. He had never suffered any injury to the œsophagus. Syphilis was denied, and he had no evidences of it.

In March, 1895, he had a severe and somewhat protracted cold. From this he recovered, but noticed, almost at once afterward, a feeling of constriction and oppression in his throat. Later he found swallowing was becoming difficult, and increasingly so. Once, upon attempting to force down with his finger a piece of meat that had caught in his throat, he vomited, and the vomit contained blood. With this